Amendments to the specification

At the indicated page and line numbers, please replace the existing paragraphs with those set forth below:

(Page 1, line 1)

~~ANTIBIOTIC PRODUCTION~~METHODS OF INCREASING ANTIBIOTIC PRODUCTION IN *STREPTOMYCES* BY DELETION OF THE *scbA* gene

(Page 12, line 34 through page 13, line 25)

"Percent (%) amino acid sequence identity" is defined as the percentage of amino acid residues in a candidate sequence that are identical with the amino acid residues in the sequence with which it is being compared, after aligning the sequences and introducing gaps, if necessary, to achieve the maximum percent sequence identity, and not considering any conservative substitutions as part of the sequence identity. The % identity values used herein are generated by WU-BLAST-2 which was obtained from Altschul et al. (1996);. Retrieved from the Internet: <Url: http://blast.wustl/edu/blast/README.html≥. WU-BLAST-2 uses several search parameters, most of which are set to the default values. The adjustable parameters are set with the following values: overlap span =1, overlap fraction = 0.125, word threshold (T) = 11. The HSPS and HSPS2 parameters are dynamic values and are established by the program itself depending upon the composition of the particular sequence and composition of the particular database against which the sequence of interest is being searched; however, the values may be adjusted to increase sensitivity. A % amino acid sequence identity value is

determined by the number of matching identical residues divided by the total number of residues of the "longer" sequence in the aligned region, multiplied by 100. The "longer" sequence is the one having the most actual residues in the aligned region (gaps introduced by WU-BLAST-2 to maximize the alignment score are ignored).

(Page 32, lines 3-24)

~~Onishi~~Ohnishi et al. (1999) reported the cascade for the streptomycin production in *S. griseus*, triggered by A-factor. *ArpA* (A-factor binding protein) binds to the promoter region of *adpA* (a transcriptional activator for streptomycin production) and represses the transcription *arpA* from the promoter region by binding to it. Thus *adpA* is transcribed and activates the streptomycin biosynthetic cluster via *strR* (streptomycin pathway-specific activator) and the antibiotic is produced. To corroborate their model, the *afsA* mutant (equivalent to the *scbA* mutant of the present work) produces neither streptomycin nor A-factor. Also the *arpA* mutant (equivalent to the *scbR* mutant of the present work) overproduces antibiotics; A-factor production is not effected. These are the reverse phenotypes compared to those of the in-frame deletion mutants of the present work using *S. coelicolor*. The inventors propose that ?-butyrolactones are involved in antibiotic production differently in *S. coelicolor*, compared with the known GBL model of *S. griseus*.